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儿童急性横贯性脊髓炎诊治进展

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摘　要

急性横贯性脊髓炎是一种以脊髓局灶性炎症为特征的脊髓疾病，虽然少见，但可能遗留严重的神经系统后遗症。近年来，随着脱髓鞘相关抗体的发现，对该病的认识也不断提高。因此，本文拟通过对急性横贯性脊髓炎相关文献的分析和总结，探讨急性横贯性脊髓炎的流行病学、发病机制、临床表现、诊断方法、鉴别诊断、治疗和预后，旨在为临床医生提供对该病的更好认识，指导临床的早期规范化诊断、针对性治疗，最终改善患者预后。

关键词

儿童，急性横贯性脊髓炎，诊治进展

Progress in the Diagnosis and Treatment of Acute Transverse Myelitis in Children

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Abstract

Acute transverse myelitis is a spinal cord disease characterized by focal inflammation of the spinal cord. Although it is rare, it may leave serious neurological sequelae. In recent years, with the discovery of demyelinating-related antibodies, the understanding of this disease has been continuously improved. Therefore, this article aims to analyze and summarize the relevant literature on

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acute transverse myelitis to explore its epidemiology, pathogenesis, clinical manifestations, diagnostic methods, differential diagnosis, treatment, and prognosis. The goal is to provide clinicians with a better understanding of the disease and guide early standardized diagnosis and targeted treatment in clinical practice, ultimately improving the prognosis of patients.

Keywords

Children, Acute Transverse Myelitis, Diagnosis and Treatment Progress

1. 引言

急性横贯性脊髓炎(acute transverse myelitis, ATM)是一种少见的以脊髓局灶性炎症为特征的脊髓疾病，临床表现为快速起病的肢体无力、感觉障碍和大小便功能障碍。ATM 可以作为一种独立的疾病，通常在前驱感染后出现，称为特发性 ATM；也可与中枢神经系统获得性脱髓鞘综合征(acquired demyelinating syndromes, ADS)相关，称为疾病相关性 ATM，这些疾病包括多发性硬化症(multiple sclerosis, MS)、视神经脊髓炎谱系疾病(neuromyelitis optica spectrum disorder, NMOSD)和髓鞘少突胶质细胞糖蛋白(myelin oligodendrocyte glyco-protein, MOG)抗体相关疾病等。本病可遗留不同程度的神经系统后遗症，严重影响患者的生活质量，故临床上需对其进行早期规范化诊断及针对性治疗，以期改善预后。

2. 流行病学

在儿童中，ATM 的每年发病率估计在 1.7/100 万~2/100 万[1]。男女比例为 1.1~1.6:1 [2]，但据报道，在多发性硬化症(multiple sclerosis, MS)或视神经脊髓炎(neuromyelitis optica, NMO)相关的青少年病例中，女性占主导地位[3]-[5]。儿童时期的发病年龄呈双峰型，分别为 5 岁以下及 10 岁以上儿童[6] [7]。没有明显的种族易感性[2]。在起病前 30 天内，66%的儿童有前驱感染史，25%的儿童有疫苗接种史[6] [8] [9]。

3. 发病机制

既往的病理研究表明大多数 ATM 患者的病理特征为病变部位血管周围有单核细胞和淋巴细胞浸润[10]，常同时存在灰白质的受累，这表明 ATM 并不是单纯的脱髓鞘疾病，而是累及神经元、轴突、少突胶质细胞和髓鞘的混合性炎症性疾病。ATM 患者病前常存在前驱感染史[11]-[17]，这提示感染相关 ATM 的发病机制可能与机体对病原体感染所产生的全身性反应有关，具体而言，分子模拟和超抗原介导可能是自身免疫的重要机制[18]。另外，有研究报道了 NMOSD 和复发性 TM 患者中自身抗体的作用机制[19]-[22]，一方面自身抗体穿过血脑屏障后激活免疫系统的其他成分；另一方面某些自身抗体可直接导致神经元或神经胶质细胞的选择性损伤，而该过程是由于这些细胞表达的抗原与抗感染性病原体抗体之间具有交叉反应[18]。

4. 临床特征

ATM 以急性或亚急性方式起病，主要表现为运动、感觉及自主神经功能障碍。神经系统症状在发病后 2~4 天内迅速进展，常在 5~6 天内达高峰[5] [6] [23]。运动症状主要表现为不同程度的肌力下降。病初查体为下运动神经元瘫痪表现(肌张力减低和腱反射减弱)，发病 2 周后开始逐步演变为上运动神经元瘫

痪表现(肌张力增加和腱反射亢进) [24]。感觉症状可以是阳性(烧灼感、感觉过敏、疼痛)或阴性(感觉缺失、麻木感)。查体常有感觉障碍平面，儿童感觉障碍平面多位于胸段[24]。感觉受累平面是疾病预后的重要因素[25]，但由于儿童查体常常不配合，故在多达 40%的儿童中，无法清晰地评估感觉障碍平面[6] [7] [26]。自主神经功能障碍包括进行性加重的尿急、大小便失禁、排尿困难或无法排尿、便秘[27]-[30]。此外，尿潴留也可能是 ATM 的首发症状[30]。95%的患者在急性期会出现尿潴留，其原因是脑桥排尿中枢和骶髓之间的信号中断[24]。

ATM 可分别根据脊髓病变横向或纵向受累的范围进行不同亚型的分类，即急性完全性横贯性脊髓炎和急性部分性横贯性脊髓炎(横向受累程度分类)，急性短节段性横贯性脊髓炎和急性长节段性横贯性脊髓炎(纵向受累程度分类)。急性完全性横贯性脊髓炎(acute complete transverse myelitis, ACTM)主要表现为病变水平以下两侧对称的运动和感觉功能障碍。急性部分性横贯性脊髓炎(acute partial transverse myelitis, APTM)的主要表现为病变水平以下两侧不对称的运动和/或感觉功能障碍。短节段性横贯性脊髓炎(short-segment transverse myelitis, STM)是指 MRI 显示病变长度为 3 个椎体节段以下的完全性或不完全性脊髓功能障碍。长节段性横贯性脊髓炎(longitudinally extensive transverse myelitis, LETM)是指 MRI 显示病变长度至少为 3 个椎体节段的完全性或不完全性脊髓功能障碍[6] [7] [26]。

5. 辅助检查

在儿童 ATM 的诊断中，除了临床特征，辅助检查也是至关重要的。常用的辅助检查包括脑脊液检查、影像学检查、脱髓鞘抗体检测等。

脑脊液检查：约有一半的患者脑脊液异常，表现为白细胞数量及蛋白水平的轻度升高，葡萄糖水平正常，部分患者脑脊液检测可有 IgG 指数升高、寡克隆带阳性。一项纳入 170 例特发性 TM 成人病例系列研究显示，脑脊液平均白细胞计数为 38/μL，平均蛋白水平 75 mg/dL [31]。但是，一项儿科病例系列研究观察到了更高的水平，平均白细胞计数为 136/μL，平均蛋白水平为 173 mg/dL (1.73 g/L) [8]。研究发现，在单症状疾病患者中，脑脊液中寡克隆带(oligoclonal bands, OCBs)阳性者发展为 MS 的风险高于寡克隆带阴性者[32] [33]。

影像学检查：脊髓 MRI 通常表现为受累节段肿胀，约 74%行钆增强扫描的患者可见 T1WI 强化信号，少数患者的脊髓 MRI 检查可以正常的[8]。因此，脊髓 MRI 检查正常并不能排除 ATM。累及脊髓节段长度通常为 1 个或多个[8] [11] [34] [35]。一项纳入 170 例特发性 TM 患者的成人病例系列研究显示[31]，病变由头侧向尾侧的累及范围可从 1 个椎体节段(多数患者)至累及全脊髓(2 例患者)。儿童中可见类似情况，病变范围平均跨越 6 个节段[8]。文献报道，66%~85%的儿童 ATM 中可见 LETM [4] [36]，这可能与 NMO 和 ADEM 相关的 TM 相关[8]。但是，MS 通常与少于三个椎体节段的节段性 TM 相关[24]。大多数脊髓病变累及颈段(64%~76%)和颈胸段[4] [8] [23]。除脊髓 MRI 病变外，有超过 40%的 ATM 患者脑部 MRI 存在无症状病变，而且此类患者发生 NMO 或 MS 的风险更高[36]。此外，有文献报道，APTM 伴脑部病变的儿童中，MS 的发生率可高达 66%~88% [7] [26]。因此，脑部 MRI 对于评估 ATM 患者是非常有必要的[7]。

此外，影像学检查对疾病相关性 ATM 的分型具有重要的鉴别意义：

1) MS-ATM：脊髓病变常见部位为颈胸髓，累及脊髓后部的多发小病灶，多位于外周白质。病变长度相对较短，通常为一到两个脊髓节段。

2) NMO-ATM：约 43%~70%的 NMOSD 患者在疾病发作时可观察到脑部病变[37]。脊髓病变主要累及胸髓，多累及中央灰质，且常表现为 LETM。

3) MOG 抗体病-ATM：2/3 的患者脑部 MRI 显示正常[38]。脊髓病变位置以中央灰质脊髓病变较为

常见，多位于胸椎节段或脊髓圆锥，可表现为轻度脊髓肿胀，呈现出“中央 H 形”脊髓病变的特征。

脱髓鞘抗体：此前关于儿童 ATM 自身抗体的研究很少，在小型的队列中，MOG 抗体阳性率为 22%~43%，AQP4 抗体阳性率为 7%~10% [4] [6] [36] [39] [40]。AQP4 抗体阳性与疾病复发相关[41]。在 MOG 抗体阳性的儿童 ATM，特别是那些 MOG 抗体持续存在的患者中，小部分患者会在随访期间复发[42]-[44]。在一项进行抗体检测的 371 名儿童的大型队列中，有 50 名患者诊断为 ATM，其中，3 例 MOG 抗体阳性(6%，均为单相)，8 例 AQP4 抗体阳性(16%，随后均被诊断为 NMOSD) [45]。

神经电生理检查：包括视觉诱发电位、体感诱发电位等。如视觉诱发电位提示异常，应注意多发性硬化、视神经脊髓炎的可能。

6. 诊断

2002 年横贯性脊髓炎协作组(Transverse Myelitis Consortium Working Group, TMCWG)制定了急性特发性脊髓炎的诊断标准[46]。但此标准在儿童 ATM 的应用具有一定的局限性。因为在儿童中无法清晰地评估感觉障碍平面[6] [7] [26]。此外，ATM 的患者中有相当大比例不符合炎症特点[47]。因此，临床诊断不一定要满足所有标准。这表明 2002 年指南在儿童 ATM 的应用需进一步研究。

在临床中，当患者出现局限于一个或多个脊柱节段的双侧运动、感觉和自主神经功能障碍的体征和症状，可考虑脊髓病变的诊断。首先需紧急行脊柱影像学检查以排除压迫性病因。再根据炎性标志物(① MRI 钆增强，或② 脑脊液细胞数增多或 IgG 指数升高)来确定炎症性或非炎症性脊髓病。如果存在炎症并且怀疑 ATM，则建议进一步检查。检查包括：脑部 MRI、寡克隆带、AQP4 抗体、MOG 抗体等检查[48]。在超过 40%的儿童中，存在无症状的脑部 MRI 病变，这是发生 MS 或 NMO 的危险因素[36]。脑脊液研究表明，寡克隆带阳性者发展为 MS 的风险增加[24]。研究表明，AQP4 抗体阳性与疾病复发相关[41]。在 MOG 抗体阳性的儿科患者中，特别是那些 MOG 抗体持续阳性的患者，小部分患者会在随访期间复发[42]-[44]。此外，建议所有患者行眼部相关检查(如视觉诱发电位)以检测是否共患病视神经炎[24]，这是因为一些患者虽然没有视力障碍的症状，但可能存在亚临床视神经炎的神经生理学表现[24]。

7. 鉴别诊断

1) 脊髓肿瘤：脊髓肿瘤通常表现为亚急性疼痛和脊髓病症状。髓外肿瘤包括神经鞘瘤、脑膜瘤和髓母细胞瘤转移。髓内肿瘤包括星形细胞瘤和室管膜瘤[2]。

2) 感染性脊髓炎：感染性脊髓炎的临床表现、脊髓 MRI 及脑脊液表现与 ATM 相似。病原体感染的证据包括脑脊液中病原体的分离、PCR 阳性结果、或急性和恢复期血清抗体滴度升高。据报道，多种病原体可引起感染性脊髓炎。肠道病毒与急性迟缓性脊髓炎有关，其典型的主要运动症状可能是由脊髓运动神经元直接感染引起的[49]。

3) 吉兰－巴雷综合征(Guillain-Barre syndrome, GBS)：本病临床表现为进展性感觉和运动功能障碍，类似于 ATM [50]。依据临床表现和辅助检查可咨鉴别。首先，GBS 患者感觉障碍相对轻微，以早期一过性主观感觉障碍为主，伴腱反射减弱或消失。其次，GBS 患者的肌电图提示为周围神经传导阻滞和/或传导延迟，但这些在 ATM 患者中通常是正常的[2]。此外，脑脊液检查可见“蛋白－细胞分离”现象，但此现象一般在起病后第 2 周出现。

8. 治疗

药物治疗是治疗 ATM 的基石，目的是减轻免疫反应，尽早地促进疾病恢复、减少后遗症的发生。

糖皮质激素：一直被作为 ATM 的一线治疗，首选方案是静脉输注甲泼尼龙(30 mg/kg，最大剂量为 1000 mg/d)冲击治疗，持续 3~5 天[2] [24] [51]。在除外禁忌证后应尽早启动糖皮质激素治疗[2]。多项研

究表明糖皮质激素在中枢神经系统炎症性疾病(包括 ATM)中的疗效和安全性。有研究表明使用糖皮质激素开始治疗的时间间隔越长与疾病预后不良相关[1] [52]。此外，研究显示，使用大剂量糖皮质激素的患者能改善短期和长期预后[51] [53]。在停用静脉甲泼尼龙后应改为泼尼松口服维持治疗，起始剂量为 1~2 mg/kg/d，并根据疾病恢复情况逐渐减停[24] [54]。

血浆置换(plasmapheresis, PLEX)：对于使用静脉激素冲击治疗后症状无明显改善或继续加重者，应考虑血浆置换[24]。当存在明显的运动或呼吸功能障碍时，一些中心会同时使用 PLEX 和糖皮质激素[55] [56]。美国神经病学学会 2011 年发布的指南中指出，有研究证明 PLEX 治疗成人 ATM 的有效性[24]。治疗方案是进行 5~7 次治疗，每次置换 1.1~1.5 倍血浆容量，隔日进行 1 次，共 10 日[57]。

静脉丙种球蛋白(intravenous immunoglobulin, IVIG)：尚不明确是否有益处[24] [26]。目前没有 IVIG 治疗 ATM 的随机对照试验。在已发表的病例系列报道中，在 IVIG 治疗后，部分患者的临床症状(如肢体力量、运动能力等)有所改善，影像学病变有所吸收[58]。IVIG 使用剂量为 2 g/kg 总量，分为 2~5 天。

免疫调节治疗：对于复发性疾病的患者，应考虑使用硫唑嘌呤、甲氨蝶呤或霉酚酸酯等药物进行长期免疫调节治疗[18]。对于诊断为 MS 或 NMO 的患者，也应在急性期治疗后使用免疫调节药物[59]。

9. 预后

9.1. 结局

与成人相比，儿童 ATM 急性期神经功能缺损虽然更严重，但其预后相对较好。恢复过程可持续数年。平均约 33%~50%的患者完全康复，仅 10%~20%的病例预后不良[24] [60]。关于疾病预后危险因素，目前仍无统一结论。研究显示，发病年龄较小、疾病达峰时间 < 24 小时、完全性瘫痪、需要辅助通气、治疗时间较长、脑脊液蛋白水平增高、感觉障碍平面较高、脊髓 MRI T1 低信号、长节段脊髓受累与预后不良相关[2] [47] [55] [61]。与特发性 ATM 和 NMO-ATM 相比，ADEM-ATM 或 MS-ATM 具有更好的恢复预后[24]。

9.2. 复发

研究表明特发性 TM 患者的复发率为 25%~33%，而疾病相关性 TM 的复发率可高达 70% [60]。与复发的相关因素包括：脊髓 MRI 示多灶性或长节段脊髓病变、MRI 显示脑部病变、脑脊液中寡克隆带阳性、MOG 抗体阳性、AQP4 抗体阳性和女性[2] [60] [62]。LETM 患者发生 NMO 的风险增加。LETM、复发性 ATM、并发或快速出现神经炎的 ATM 提示 NMOSD [24]。有脑部 MRI 病灶、脑脊液寡克隆带或者 IgG 指数升高可能是 MS 的高危因素[63]。

10. 总结

儿童 ATM 是一种少见的以脊髓局灶性炎症为特征的脊髓疾病，临床表现为快速起病的肢体无力、感觉和大小便功能障碍。它可以作为一种独立的疾病，通常为前驱感染后出现，称为特发性 ATM；也可与 ADS 相关，称为疾病相关性 ATM。诊断 ATM 需要排除压迫性脊髓病变(一般通过脊髓 MRI)，并通过 MRI 钆增强或腰椎穿刺证实炎症存在。建议静脉用大剂量糖皮质激素来治疗 ATM，如果症状没有改善，可考虑血浆置换或 IVIG 治疗。儿童 ATM 的预后虽然较成人更好，但仍有一部分儿童可能遗留严重的神经系统后遗症。

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